STIC-FPAS

From: Harrison, Robert
To: STIC-FPAS
Subject: 08/396,446 ; Pat.
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Examiner	: Robert H. Harrison
Art Unit	: 1501
Office Location	: Crystal Mall One, Room 5E09
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U.S. Serial No.	: 08/396,446
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Date of request	: 6/24/96
Date Needed	: Normal Priority

Patent: JP 07112923 A2
Date: May 2, 1995 (Heisei)
Title: Hair growth-stimulating compositions...

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MEDLINE EXPRESS (R) 1/96-2/96 usage is subject to the terms and conditions of the Subscription and License Agreement and the applicable Copyright and intellectual property protection as dictated by the appropriate laws of your country and/or by International Convention.

TI: Mycophenolic acid, an inhibitor of IMP dehydrogenase that is also an immunosuppressive agent, suppresses the cytokine-induced nitric oxide production in mouse and rat vascular endothelial cells.
AU: Senda-M; DeLustro-B; Eugui-E; Natsumeda-Y
AD: Institute of Biochemistry and Cell Biology, Syntex Discovery Research, Palo Alto, California 94303, USA.
SO: Transplantation. 1995 Nov 27; 60(10): 1143-8
ISSN: 0041-1337
PY: 1995
LA: ENGLISH
CP: UNITED-STATES
AB: Mycophenolic acid (MPA), an inhibitor of IMP dehydrogenase and de novo GTP biosynthesis, also has immunosuppressive activity. The effect of MPA on nitric oxide (NO) production by rodent brain vascular endothelial cells in culture was investigated. MPA inhibited NO production by mouse and rat brain endothelial cells that had been stimulated with a combination of interferon-gamma and tumor necrosis factor-alpha. The 50% inhibitory concentration (EC50) was in the range of 0.5-1.0 microM. However, MPA had no effect on basal NO production in mouse brain vascular endothelial cells. Brequinar, an inhibitor of de novo pyrimidine synthesis, had no effect on NO production in cytokine stimulated endothelial cells. Guanosine, which can act as a salvage pathway precursor for GTP biosynthesis, reversed the inhibitory effect of MPA in a dose-dependent fashion. We suggest that inducible NO synthase activity is dependent on GTP level and can be blocked by curtailing IMP dehydrogenase activity.
MESH: Cells,-Cultured; Guanosine-pharmacology; Mice-; Pteridines-pharmacology; Rats-
MESH: *Cytokines-pharmacology; *Endothelium,-Vascular-metabolism; *Immunosuppressive-Agents-pharmacology; *IMP-Dehydrogenase-antagonists-and-inhibitors; *Mycophenolic-Acid-pharmacology; *Nitric-Oxide-biosynthesis
TG: Animal; Support,-U.S.-Gov't,-P.H.S.
PT: JOURNAL-ARTICLE
CN: 1R55CA6034401CANCI
RN: EC 1.1.1.205; 0; 0; 0; 10102-43-9; 118-00-3; 17094-01-8; 24280-93-1
NM: IMP-Dehydrogenase; Cytokines; Immunosuppressive-Agents; Pteridines; Nitric-Oxide; Guanosine; sepiapterin; Mycophenolic-Acid
AN: 96083863
UD: 9602

TI: Mycophenolate mofetil (RS 61443): nothing new under the sun or an important break-through in the field of transplantation? [editorial]
AU: van-der-Woude-FJ
SO: Nephrol-Dial-Transplant. 1995; 10(7): 1112-5
ISSN: 0931-0509
PY: 1995
LA: ENGLISH
CP: ENGLAND
MESH: Cell-Division-drug-effects; Lymphocytes-cytology; Lymphocytes-drug-effects; Mycophenolic-Acid-pharmacology; Mycophenolic-Acid-therapeutic-use

MESH: *Immunosuppressive-Agents-therapeutic-use;

*Kidney-Transplantation-trends; *Mycophenolic-Acid-analogs-and-derivatives
TG: Animal; Human
PT: EDITORIAL; REVIEW; REVIEW,-TUTORIAL
RN: 0; 128794-94-5; 24280-93-1
NM: Immunosuppressive-Agents; RS-61443; Mycophenolic-Acid
AN: 96000678
UD: 9602

TI: Reversible modulation of opioid receptor binding in intact neural cells by endogenous guanosine triphosphate.
AU: Yabaluri-N; Medzihradsky-F
AD: Department of Biological Chemistry, University of Michigan Medical School, Ann Arbor 48109, USA.
SO: Mol-Pharmacol. 1995 Oct; 48(4): 690-5
ISSN: 0026-895X
PY: 1995
LA: ENGLISH
CP: UNITED-STATES
AB: Incubation of SH-SY5Y neural cells with mycophenolic acid (MPA), an inhibitor of inosine monophosphate dehydrogenase (the key enzyme in purine nucleotide biosynthesis), reduced the cellular content of GTP by 94% relative to its concentration in control cells (43 nmol/mg protein) without altering the level of GDP. Although in GTP-depleted intact cells the receptor binding parameters (Kd and Bmax) of the opioid antagonist [3H]naltrexone were unchanged from those in untreated cells, the binding affinity of the mu-selective opioid agonist [3H]Tyr-D-Ala-Gly-(Me)- Phe-Gly-ol ([3H]DAMGO) was enhanced 2-fold. Furthermore, the kinetics of ligand/receptor interaction revealed that in the nucleotide-depleted cells, the dissociation rate constant for [3H]DAMGO was reduced by 44%. Initial exposure of SH-SY5Y cells to pertussis toxin reduced high-affinity ligand binding by 95% and abolished the effect of MPA treatment. Renewed incubation of the GTP-depleted cells with guanosine restored the original GTP levels and agonist binding. Neither MPA nor guanosine treatment changed the Bmax of [3H]DAMGO binding. Forskolin- and prostaglandin E1-stimulated adenylyl cyclase activities were decreased significantly in GTP-depleted cells. DAMGO and [D-Pen2,D-Pen5]enkephalin inhibitions of adenylyl cyclase were also affected with MPA treatment. Maximal inhibition of forskolin-stimulated adenylyl cyclase activity by both of the agonists was reduced, whereas MPA caused a 2-fold reduction in potency for DAMGO. The results show that reduction in endogenous GTP levels leads to noticeable changes in agonist, receptor, and G protein interactions, as measured by agonist binding, and to subsequent diminution of the signal transduction, as reflected by the cAMP levels.
MESH: Adenyl-Cyclase-analysis; Amino-Acid-Sequence; Analgesics-metabolism; Analgesics-pharmacology; Enkephalins-metabolism; Enkephalins-pharmacology; G-Proteins-metabolism; Guanosine-Triphosphate-metabolism; Kinetics-; Molecular-Sequence-Data; Mycophenolic-Acid-pharmacology; Naltrexone-metabolism; Narcotic-Antagonists-metabolism; Neuroblastoma-; Neurons-physiology; Neurons-ultrastructure; Receptors,-Opioid,-mu-agonists; Receptors,-Opioid,-mu-antagonists-and-inhibitors; Tritium-; Tumor-Cells,-Cultured
MESH: *Guanosine-Triphosphate-physiology; *Neurons-metabolism; *Receptors,-Opioid,-mu-metabolism
TG: Human; Support,-U.S.-Gov't,-P.H.S.
PT: JOURNAL-ARTICLE
CN: DA00254DANIDA
RN: EC 4.6.1.1; 0; 0; 0; 0; 0; 10028-17-8; 16590-41-3; 24280-93-1; 78123-71-4; 86-01-1
NM: Adenyl-Cyclase; Analgesics; Enkephalins; G-Proteins; Narcotic-Antagonists; Receptors,-Opioid,-mu; Tritium; Naltrexone; Mycophenolic-Acid;

enkephalin,-Ala(2)-MePhe(4)-Gly(5)-; Guanosine-Triphosphate

AN: 96029779
UD: 9602

TI **Hair** growth-stimulating compositions containing **mycophenolic** acid or its derivatives
IN Tamura, Gakuzo; Ando, Kunio; Nakamura, Tetsuo
PA Imuno Japan Kk, Japan
SO Jpn. Kokai Tokkyo Koho, 5 pp.
CODEN: JKXXAF
PI JP 07112923 A2 950502 Heisei
AI JP 93-289710 931015
DT Patent
LA Japanese
IC ICM A61K007-06
ICS A61K031-365
ICA C07D307-88
CC 62-3 (Essential Oils and Cosmetics)
OS MARPAT 123:92871
GI

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TI: Anti-angiogenesis: a multipurpose therapeutic tool? [editorial]
AU: Ribatti-D; Vacca-A; Bertossi-M; Roncali-L
SO: Int-J-Clin-Lab-Res. 1993; 23(3): 117-20
ISSN: 0940-5437
PY: 1993
LA: ENGLISH
CP: GERMANY
MESH: *Angiogenesis-Factor-antagonists-and-inhibitors;
*Neovascularization,-Pathologic-therapy
TG: Human
PT: EDITORIAL
RN: 0
NM: Angiogenesis-Factor
AN: 94003361
UD: 9401

MEDLINE EXPRESS (R) 1990 usage is subject to the terms and conditions of the Subscription and License Agreement and the applicable Copyright and intellectual property protection as dictated by the appropriate laws of your country and/or by International Convention.

TI: Angiogenic activity is defective in monocytes from patients with alopecia universalis.
AU: Skoutelis-A; Freinkel-RK; Kaufman-DS; Leibovich-SJ
AD: Department of Dermatology, Northwestern University Medical School, Chicago, IL 60611.
SO: J-Invest-Dermatol. 1990 Aug; 95(2): 139-43
ISSN: 0022-202X
PY: 1990
LA: ENGLISH
CP: UNITED-STATES
AB: Monocyte/macrophages are important components of cell-mediated immune responses in presentation of antigen, as regulators of lymphocyte function, and as sources of cytokines that modulate functions of cells other than those of the immune system. Their role in the pathogenesis of alopecia areata (AA) and universalis (AU) has not been explored. This study is an investigation of the function of peripheral blood monocytes from normal subjects and patients with AA, AU, and alopecia totalis (AT), with respect to the principal macrophage-derived angiogenic factor, tumor necrosis factor alpha (TNF alpha). Because neovascularization is a necessary component in the anagen phase of hair growth and may play a role in the pathology of these disorders, we asked whether monocyte/macrophage angiogenic activity was compromised in these alopecias. Purified preparations of monocytes were activated in culture. Conditioned media were assessed for angiogenic activity on the chick chorioallantoic membrane and for concentration of TNF alpha by enzyme-linked immunosorbent assay (ELISA). Both angiogenic and the TNF concentration were significantly diminished in conditioned media from AU monocytes when compared to those from normal subjects and patients with AA. These results show that the function of AU monocytes may be abnormal and that the abnormality may distinguish AU from AA. Defective monocyte/macrophage function could also play a pathogenic role via effects on neovascularization and/or modulation of the immune response.
MESH: Adult-; Allantois-; Alopecia-blood; Cells,-Cultured; Chick-Embryo; Chorion-; Enzyme-Linked-Immunosorbent-Assay; Reference-Values; Tumor-Necrosis-Factor-analysis
MESH: *Alopecia-physiopathology; *Monocytes-physiology; *Neovascularization,-Pathologic
TG: Animal; Human
PT: JOURNAL-ARTICLE
RN: 0
NM: Tumor-Necrosis-Factor
AN: 90338793
UD: 9011

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Marked in Search: #30

TI: Mycophenolic acid and some antioxidants induce differentiation of monocytic lineage cells and augment production of the IL-1 receptor antagonist.
AU: Waters-RV; Webster-D; Allison-AC
AD: Syntex Discovery Research, Palo Alto, California 94304.
SO: Ann-N-Y-Acad-Sci. 1993 Nov 30; 696: 185-96
ISSN: 0077-8923
PY: 1993
LA: ENGLISH
CP: UNITED-STATES
MESH: Cell-Differentiation-drug-effects; Cell-Division-drug-effects; Monocytes-cytology; Tumor-Cells,-Cultured
MESH: *Antioxidants-pharmacology; *Interleukin-1-antagonists-and-inhibitors; *Monocytes-drug-effects; *Mycophenolic-Acid-pharmacology; *Sialoglycoproteins-biosynthesis
TG: Human
PT: JOURNAL-ARTICLE
RN: 0; 0; 0; 0; 24280-93-1
NM: interleukin-1-receptor-antagonist-protein; Antioxidants; Interleukin-1; Sialoglycoproteins; Mycophenolic-Acid
AN: 94152915
UD: 9405

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TI: Keratinocyte growth factor is an important endogenous mediator of hair follicle growth, development, and differentiation. Normalization of the nu/nu follicular differentiation defect and amelioration of chemotherapy-induced alopecia.

AU: Danilenko-DM; Ring-BD; Yanagihara-D; Benson-W; Wiemann-B; Starnes-CO; Pierce-GF
AD: Department of Experimental Pathology, Amgen Inc., Thousand Oaks, California 91320-1789, USA.

SO: Am-J-Pathol. 1995 Jul; 147(1): 145-54
ISSN: 0002-9440
PY: 1995
LA: ENGLISH
CP: UNITED-STATES

AB: The growth and development of hair follicles is influenced by a number of different growth factors and cytokines, particularly members of the fibroblast growth factor (FGF) family. Keratinocyte growth factor (KGF or FGF-7) is a recently identified 28-kd member of the FGF family that induces proliferation of a wide variety of epithelial cells, including keratinocytes within the epidermis and dermal adnexa. Because KGF induces marked proliferation of keratinocytes, and both KGF and KGF receptor (KGFR) mRNA are expressed at high levels in skin, we sought to localize KGF and KGFR in skin by in situ hybridization. KGFR mRNA was relatively strongly expressed by keratinocytes in the basilar epidermis as well as throughout developing hair follicles of rat embryos and neonates. KGF mRNA was expressed at lower levels than was KGFR but could be localized to follicular dermal papillae in rat embryos and neonates. These results prompted us to investigate the effects of KGF on hair follicles in two distinct murine models of alopecia. In the first model, recombinant KGF (rKGF) induced dose-dependent hair growth over most of the body in nu/nu athymic nude mice when administered intraperitoneally or subcutaneously over 17 to 18 days. When administered subcutaneously, rKGF induced the most extensive hair growth at the sites of injection. Histologically, rKGF induced marked follicular and sebaceous gland hypertrophy, a normalization of the nu/nu follicular keratinization defect, and an increase in follicular keratinocyte proliferation as assessed by bromodeoxyuridine labeling. In the second model, a neonatal rat model of cytosine arabinoside chemotherapy-induced alopecia in which interleukin-1, epidermal growth factor, and acidic FGF have all demonstrated some degree of alopecia cytoprotection, rKGF induced a dose-dependent cytoprotective effect, abrogating as much as 50% of the alopecia in this model when administered beginning 1 day before the onset of chemotherapy. Taken together, these data suggest that KGF is an important endogenous mediator of normal hair follicle growth, development, and differentiation.

MESH: Alopecia-chemically-induced; Alopecia-pathology; Animals,-Newborn; Cell-Differentiation-drug-effects; Cell-Division-drug-effects; Cytarabine-; Dose-Response-Relationship,-Drug; Growth-Substances-biosynthesis; Growth-Substances-pharmacology; Hair-drug-effects; In-Situ-Hybridization; Keratinocytes-cytology; Keratinocytes-drug-effects; Mice-; Mice,-Nude; Pregnancy-; Rats-; Rats,-Sprague-Dawley; Receptors,-Growth-Factor-biosynthesis; Recombinant-Proteins; RNA,-Messenger-biosynthesis; Sebaceous-Glands-cytology; Sebaceous-Glands-drug-effects; Skin-drug-effects; Skin-metabolism;

Skin-pathology
MESH: *Alopecia-prevention-and-control; *Growth-Substances-physiology; *Hair-cytology; *Hair-growth-and-development
TG: Animal; Female
PT: JOURNAL-ARTICLE
RN: 0; 0; 0; 0; 0; 126469-10-1; 147-94-4
NM: keratinocyte-growth-factor-receptor; Growth-Substances; Receptors,-Growth-Factor; Recombinant-Proteins; RNA,-Messenger; keratinocyte-growth-factor; Cytarabine
AN: 95328610
UD: 9510
SB: AIM

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Marked in Search: #40

TI: Cultured human hair follicles and growth factors.
AU: Philpott-MP; Sanders-D; Kealey-T
AD: Department of Clinical Biochemistry, University of Cambridge, Addenbrookes Hospital, United Kingdom.
SO: J-Invest-Dermatol. 1995 May; 104(5 Suppl): 44S-45S
ISSN: 0022-202X
PY: 1995
LA: ENGLISH
CP: UNITED-STATES
MESH: Alopecia-Areata-chemically-induced; Alopecia-Areata-physiopathology; Cell-Division-drug-effects; Culture-Media,-Serum-Free; Epidermal-Growth-Factor-Urogastrone-pharmacology; Growth-Substances-pharmacology; Hair-drug-effects; Insulin-Like-Growth-Factor-I-pharmacology; Insulin-Like-Growth-Factor-II-pharmacology; Interleukin-1-pharmacology; Tissue-Culture; Tumor-Necrosis-Factor-pharmacology
MESH: *Growth-Substances-physiology; *Hair-growth-and-development
TG: Human; Support,-Non-U.S.-Gov't
PT: JOURNAL-ARTICLE
RN: 0; 0; 0; 0; 62229-50-9; 67763-96-6; 67763-97-7
NM: Culture-Media,-Serum-Free; Growth-Substances; Interleukin-1; Tumor-Necrosis-Factor; Epidermal-Growth-Factor-Urogastrone; Insulin-Like-Growth-Factor-I; Insulin-Like-Growth-Factor-II
AN: 95256686
UD: 9508

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TI: Hair growth-stimulating effects of cyclosporin A and FK506, potent immunosuppressants.
AU: Yamamoto-S; Kato-R
AD: Department of Pharmacology, School of Medicine, Keio University, Tokyo, Japan.
SO: J-Dermatol-Sci. 1994 Jul; 7 Suppl: S47-54
ISSN: 0923-1811
PY: 1994
LA: ENGLISH
CP: IRELAND
AB: Cyclosporin A (CsA), a cyclic endecapeptide, is a T cell-specific immunosuppressant and is successfully used in the field of organ transplantation. Another T cell-specific immunosuppressant, FK506, a more recently discovered macrolide antibiotic, is effective against graft rejection at much lower doses than CsA. Although totally different in structure, both compounds inhibit T cell activation by interfering with the production of interleukin-2 (IL-2) by inhibiting IL-2 gene expression, probably through the inhibition of calcineurin, a Ca2+/calmodulin-dependent phosphatase. Clinical studies have revealed that FK506 induces a variety of side effects in common with CsA. One of the most common side effects of CsA is hypertrichosis. The hair growth stimulating effect of CsA is observed not only in normal but also in pathological conditions of hair growth, i.e. in patients with alopecia areata and also in some patients with male-pattern alopecia. Although hypertrichosis is induced by both topical and oral administration of CsA, there has been no report showing that FK506 induces hypertrichosis. Recently we have found that topical application of FK506 to skins of mice, rats and hamsters markedly stimulates hair growth. This hair growth stimulating effect of FK506 is observed when applied topically but not by oral administration, even with a dose which causes marked immunosuppression. The hair growth stimulating effect of FK506 in normal animals may apparently be unrelated to its immunosuppressive effect. In vitro studies revealed that FK506 directly stimulates hair follicles. Mechanisms of hair growth stimulating effects of FK506 and CsA remain to be elucidated.(ABSTRACT TRUNCATED AT 250 WORDS)
MESH: Cyclosporine-administration-and-dosage; Cyclosporine-adverse-effects; Hypertrichosis-chemically-induced; Immunosuppressive-Agents-pharmacology; Tacrolimus-administration-and-dosage; Tacrolimus-adverse-effects
MESH: *Cyclosporine-pharmacology; *Hair-drug-effects; *Hair-growth-and-development; *Tacrolimus-pharmacology
TG: Animal; Female; Human; Male
PT: JOURNAL-ARTICLE; REVIEW; REVIEW,-TUTORIAL
RN: 0; 109581-93-3; 59865-13-3
NM: Immunosuppressive-Agents; Tacrolimus; Cyclosporine
AN: 95092662
UD: 9503

MEDLINE EXPRESS (R) 1991-1995 usage is subject to the terms and conditions of the Subscription and License Agreement and the applicable Copyright and intellectual property protection as dictated by the appropriate laws of your country and/or by International Convention.

TI: IL-1 alpha inhibits human hair follicle growth and hair fiber production in whole-organ cultures.
AU: Harmon-CS; Nevins-TD
AD: Hoffmann-La Roche Inc., Nutley, NJ 07110.
SO: Lymphokine-Cytokine-Res. 1993 Aug; 12(4): 197-203
ISSN: 1056-5477
PY: 1993
LA: ENGLISH
CP: UNITED-STATES
AB: In this study we have used a recently developed human hair follicle whole-organ culture system to investigate the effect of IL-1 alpha on hair follicle growth and hair fiber production. In the presence of 10 ng/ml IL-1 alpha, the growth of cultured human hair follicles ceased within 2-4 days, whereas control hair follicles grew for a period of 7-10 days. IL-1 alpha also inhibited hair fiber growth, but with an onset which occurred 3 days later than that of follicle growth inhibition. An IC50 value of approximately 30 pg/ml was obtained for IL-1 alpha inhibition of follicle growth. Incubation of hair follicles with IL-1 alpha resulted in a rapid, transient reduction in the rate of whole-follicle DNA synthesis. 1000-fold molar excess of IL-1 receptor antagonist prevented IL-1-induced follicle growth inhibition, while antagonist alone was without effect. The selective PKC inhibitor, Ro 31-7549, augmented IL-1-induced inhibition of hair follicle growth, but did not itself affect hair follicle growth. These findings indicate that IL-1 alpha exerts a rapid antiproliferative effect on hair follicles, and that inhibition of hair fiber growth is a secondary response. Thus, IL-1 may play a role in the pathophysiology of inflammatory hair loss conditions, such as alopecia areata, through a direct growth-inhibitory effect on hair follicles.
MESH: Cell-Division-drug-effects; Dose-Response-Relationship,-Drug; DNA-biosynthesis; Hair-cytology; Hair-growth-and-development; Kinetics-; Organ-Culture; Recombinant-Proteins-pharmacology; Thymidine-metabolism; Time-Factors
MESH: *Hair-drug-effects; *Interleukin-1-pharmacology
TG: Human
PT: JOURNAL-ARTICLE
RN: 0; 0; 50-89-5; 9007-49-2
NM: Interleukin-1; Recombinant-Proteins; Thymidine; DNA
AN: 94032761
UD: 9402

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TI: IL-1 alpha inhibits human hair follicle growth and hair fiber production in whole-organ cultures.
AU: Harmon-CS; Nevins-TD
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SO: Lymphokine-Cytokine-Res. 1993 Aug; 12(4): 197-203
ISSN: 1056-5477
PY: 1993
LA: ENGLISH
CP: UNITED-STATES
AB: In this study we have used a recently developed human hair follicle whole-organ culture system to investigate the effect of IL-1 alpha on hair follicle growth and hair fiber production. In the presence of 10 ng/ml IL-1 alpha, the growth of cultured human hair follicles ceased within 2-4 days, whereas control hair follicles grew for a period of 7-10 days. IL-1 alpha also inhibited hair fiber growth, but with an onset which occurred 3 days later than that of follicle growth inhibition. An IC50 value of approximately 30 pg/ml was obtained for IL-1 alpha inhibition of follicle growth. Incubation of hair follicles with IL-1 alpha resulted in a rapid, transient reduction in the rate of whole-follicle DNA synthesis. 1000-fold molar excess of IL-1 receptor antagonist prevented IL-1-induced follicle growth inhibition, while antagonist alone was without effect. The selective PKC inhibitor, Ro 31-7549, augmented IL-1-induced inhibition of hair follicle growth, but did not itself affect hair follicle growth. These findings indicate that IL-1 alpha exerts a rapid antiproliferative effect on hair follicles, and that inhibition of hair fiber growth is a secondary response. Thus, IL-1 may play a role in the pathophysiology of inflammatory hair loss conditions, such as alopecia areata, through a direct growth-inhibitory effect on hair follicles.
MESH: Cell-Division-drug-effects; Dose-Response-Relationship,-Drug; DNA-biosynthesis; Hair-cytology; Hair-growth-and-development; Kinetics-; Organ-Culture; Recombinant-Proteins-pharmacology; Thymidine-metabolism; Time-Factors
MESH: *Hair-drug-effects; *Interleukin-1-pharmacology
TG: Human
PT: JOURNAL-ARTICLE
RN: 0; 0; 50-89-5; 9007-49-2
NM: Interleukin-1; Recombinant-Proteins; Thymidine; DNA
AN: 94032761
UD: 9402

DERWENT-ACC-NO: 1998-209502

DERWENT-WEEK: 199819

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TITLE:
Compositions for retarding or inhibiting hair growth - containing sulpho:transferase inhibitor, for cosmetic or pharmaceutical use, e.g. in treatment of hirsutism

INVENTOR:DURANTON, A

PATENT-ASSIGNEE: L'OREAL SA[OREA]

PRIORITY-DATA: 1996FR-0011319 (September 17, 1996)

PATENT-FAMILY:

PUB-NO	PUB-DATE	LANGUAGE	PAGES	MAIN-IPC
FR 2753375 A1	March 20, 1998	N/A	015	A61K 031/06

APPLICATION-DATA:

PUB-NO	APPL-DESCRIPTOR	APPL-NO	APPL-DATE
FR 2753375A1	N/A	1996FR-0011319	September 17, 1996

IPC: A61K007/06; A61K031/06 ; A61K031/19

ABSTRACTED-PUB-NO:FR 2753375A
BASIC-ABSTRACT:The use of a sulphotransferase inhibitor (I) is claimed in a cosmetic composition, or in the preparation of a medicament, for retarding or inhibiting the growth of hair. Also claimed are: (I)-containing compositions for use as above; products for use as above, containing (I) and another active agent for simultaneous, separate or sequential use; and corresponding treatment methods. USE - (I), on systemic or preferably topical administration, retards or inhibits the growth of hair, and is useful for treating various skin and scalp disorders, especially hirsutism. Formulations include lotions, shampoos, tablets, syrups or nutritional supplements. ADVANTAGE - A small amount of (I) has a marked effect on hair growth on application once or twice daily.

CHOSEN-DRAWING:Dwg.0/0

TITLE-TERMS:
COMPOSITION RETARD INHIBIT HAIR GROWTH CONTAIN SULPHO TRANSFERASE INHIBIT
COSMETIC PHARMACEUTICAL TREAT HIRSUTISM

DERWENT-CLASS: B05 D21

CPI-CODES: B04-B03B; B05-A01B; B05-C01; B07-D11; B10-C02; B10-C04C; B10-D01; B10-E02; B10-E04B; B14-R02; D08-B07; D08-B09;

CHEMICAL-CODES:
Chemical Indexing M2 *01*
Fragmentation Code
C017 C100 C500 C730 C801 C804 C806 C807 M411 M781
M903 M904 M910 P616 P930 P943 Q252 V812
Specfic Compounds
01947U
Registry Numbers
1947U

Chemical Indexing M2 *02*
Fragmentation Code
G010 G020 G021 G040 G100 G221 J0- J011 J1 J131
M280 M320 M414 M510 M520 M531 M540 M781 M903 M904